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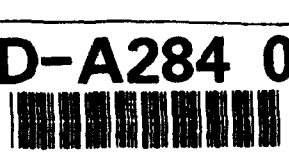

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Short Report

Hepatitis E virus in Indonesia

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Hepatitis E virus (HEV) has been identified as a major cause of enterically transmitted non-A, non-B hepatitis (ET-NANBH) (BRADLEY, 1992). The geographical distribution of HEV transmission in south-east Asia is unknown, although transmission has been reported in Hong Kong (LOK *et al.*, 1992), and HEV complementary deoxyribonucleic acid has been identified in faecal material from patients with acute hepatitis living in West Kalimantan (formerly Borneo), Indonesia (REYES *et al.*, 1990). The West Kalimantan samples were collected during an ET-NANBH outbreak in 1987 that was reported to have affected 2000 Indonesians (BRADLEY, 1992). Since that time, similar ET-NANBH outbreaks have been reported from the same area in 1989 and 1991 (I. Lubis, unpublished data). HEV may also be common on other islands in the Indonesian archipelago (RUSSELL, 1990). Using a recently developed enzyme immunoassay (EIA) for antibody to hepatitis E virus (anti-HEV) (GOLDSMITH *et al.*, 1992), we evaluated recurrent HEV transmission in West Kalimantan, Indonesia.

Eighty-nine serum samples from patients with acute hepatitis during the 1991 outbreak were analysed. All sera were tested for anti-hepatitis A virus immunoglobulin M (anti-HAV IgM), hepatitis B surface antigen (HBsAg) and anti-hepatitis core antigen (anti-HBc) IgM by EIA (Abbott Laboratories, Abbott Park, Illinois, USA). Sera were also tested by EIA (Diagnostic Biotechnology, Singapore) for antibody to hepatitis C virus (anti-HCV). A commercially-available EIA (kindly supplied by L. Chan, Diagnostic Biotechnology) was used to detect total anti-HEV, based on previously described methods (LOK *et al.*, 1992). To evaluate HEV infection further, 30 serum samples were 'blindly' assayed for anti-HEV IgM and immunoglobulin G (IgG) by Western blotting, as described previously (HYAMS *et al.*, 1992).

All 89 sera from patients with acute hepatitis were negative for anti-HAV IgM and anti-HBc IgM, but 21 (23%) had HBsAg without anti-HBc IgM and 2 (2%) had anti-HCV. The sera of 79 (88%) patients were reactive for anti-HEV by EIA. Western blot analysis of 25 EIA positive samples showed that 8 (27%) were positive for both IgM and IgG anti-HEV, and 3 (10%) were positive for IgG alone. All 5 of the samples negative by EIA for anti-HEV were also negative by Western blotting.

Both an epidemic and sporadic endemic form of HEV transmission have been described (BRADLEY, 1992). The current investigation indicates that repeated outbreaks of HEV infection can also occur in the same geographical area. A more thorough investigation is planned to determine the source of recurrent HEV transmission in West Kalimantan, Indonesia, and to determine the degree of immunity after HEV infection and whether acute clinical hepatitis E can occur more than once in the same individual. The ease and availability of the EIA for anti-HEV will be of great benefit for the diagnosis of acute hepatitis E when combined with clinical findings and the absence of other hepatitis markers. The reason why a higher percentage of serum samples were positive for anti-HEV by EIA than by Western blotting will require further investigation.

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